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=> s nf kappa beta

L1          81 NF KAPPA BETA

=> s nf kb

L2        1510 NF KB

=> s nf kappa b

L3       19775 NF KAPPA B

=> s l1 or l2 or l3

L4       20599 L1 OR L2 OR L3

=> s l4 and (antagonist or decoy or aptamer)

L5         402 L4 AND (ANTAGONIST OR DECOY OR APTAMER)

=> dup rem l5

PROCESSING COMPLETED FOR L5
L6          178 DUP REM L5 (224 DUPLICATES REMOVED)

=> s l6 and oligonucleotide#

L7           24 L6 AND OLIGONUCLEOTIDE#

=> d 1-24 ti


L7   ANSWER 1 OF 24  MEDLINE
TI   Use of phosphorothioate-modified oligodeoxynucleotides to inhibit
     NF-kappaB expression and lymphocyte function.

L7   ANSWER 2 OF 24  MEDLINE
TI   A novel strategy for myocardial protection using in vivo
     transfection of cis element 'decoy' against NFkappaB
     binding site: evidence for a role of NFkappaB in
     ischemia-reperfusion injury.

L7   ANSWER 3 OF 24  MEDLINE
TI   Controversies in the cellular pharmacology of oligodeoxynucleotides.

L7   ANSWER 4 OF 24  MEDLINE
TI   In vivo transfection of cis element "decoy" against
     nuclear factor-kappaB binding site prevents myocardial infarction
     [see comments].

L7   ANSWER 5 OF 24  MEDLINE
TI   Hypoxia induces cyclooxygenase-2 via the NF-kappaB p65 transcription
     factor in human vascular endothelial cells.

L7   ANSWER 6 OF 24  MEDLINE
TI   Manipulation of distinct NFkappaB proteins alters
     interleukin-1beta-induced human rheumatoid synovial fibroblast
     prostaglandin E2 formation.

L7   ANSWER 7 OF 24  MEDLINE
TI   Induction of neuroprotective kappa B-dependent transcription by
     secreted forms of the Alzheimer's beta-amyloid precursor.

L7   ANSWER 8 OF 24  MEDLINE
TI   The NF-kappaB transcription factor in oncogenesis.
```

L7 ANSWER 9 OF 24 MEDLINE
TI Transcription factor **decoy** approach to decipher the role of **NF-kappa B** in oncogenesis.

L7 ANSWER 10 OF 24 MEDLINE
TI Sequence-specific interaction of alpha-beta-anomeric double-stranded DNA with the p50 subunit of **NF kappa B** : application to the **decoy** approach.

L7 ANSWER 11 OF 24 MEDLINE
TI Interleukin 1 induces expression of the human immunodeficiency virus alone and in synergy with interleukin 6 in chronically infected U1 cells: inhibition of inductive effects by the interleukin 1 receptor **antagonist**.

L7 ANSWER 12 OF 24 MEDLINE
TI Inhibition of phorbol ester-induced cellular adhesion by competitive binding of **NF-kappa B** in vivo.

L7 ANSWER 13 OF 24 MEDLINE
TI Aurothioglucose inhibits induced **NF-kB** and AP-1 activity by acting as an IL-1 functional **antagonist**.

L7 ANSWER 14 OF 24 MEDLINE
TI Interleukin 1 induces **NF-kappa B** through its type I but not its type II receptor in lymphocytes.

L7 ANSWER 15 OF 24 BIOSIS COPYRIGHT 1998 BIOSIS
TI Neuroprotection by dehydroepiandrosterone-sulfate: Role of an NFkB-like factor.

L7 ANSWER 16 OF 24 BIOSIS COPYRIGHT 1998 BIOSIS
TI Feasibility of **decoy** strategy by targeting the transcription factor **NF-kappa-B** in anti-GBM nephritis.

L7 ANSWER 17 OF 24 BIOSIS COPYRIGHT 1998 BIOSIS
TI Thiol agents and Bcl-2 identify an alphavirus-induced apoptotic pathway that requires activation of the transcription factor **NF-kappa B**.

L7 ANSWER 18 OF 24 BIOSIS COPYRIGHT 1998 BIOSIS
TI Inhibition of human mesangial cell proliferation by **decoy oligonucleotide** targeting the transcription factor, **NF-kappa-B**.

L7 ANSWER 19 OF 24 CAPLUS COPYRIGHT 1998 ACS
TI Remedy and preventive for diseases caused by **NF-**. **kappa.B**

L7 ANSWER 20 OF 24 CAPLUS COPYRIGHT 1998 ACS
TI Gene therapy of renal diseases

L7 ANSWER 21 OF 24 CAPLUS COPYRIGHT 1998 ACS
TI In vivo therapeutic use of **oligonucleotide** cis-element decoys for transcription factor binding

L7 ANSWER 22 OF 24 SCISEARCH COPYRIGHT 1998 ISI (R)
TI Antisense **oligonucleotide** therapeutics for human leukemia

L7 ANSWER 23 OF 24 SCISEARCH COPYRIGHT 1998 ISI (R)
TI A 361 base pair region of the rat FSH-beta promoter contains multiple progesterone receptor-binding sequences and confers progesterone responsiveness

L7 ANSWER 24 OF 24 [redacted]CISEARCH COPYRIGHT 1998 ISI ([redacted]
TI IL-4-INDUCED EXPRESSION OF THE IL-1 RECEPTOR **ANTAGONIST** GENE IS MEDIATED BY STAT6

=> d 1 2 3 4 8 9 12 16 18 20 21

L7 ANSWER 1 OF 24 MEDLINE
AN 1998141999 MEDLINE
DN 98141999
TI Use of phosphorothioate-modified oligodeoxynucleotides to inhibit NF-kappaB expression and lymphocyte function.
AU Khaled A R; Butfiloski E J; Sobel E S; Schiffenbauer J
CS Division of Rheumatology and Clinical Immunology, University of Florida, Gainesville, Florida 32620, USA.
SO CLINICAL IMMUNOLOGY AND IMMUNOPATHOLOGY, (1998 Feb) 86 (2) 170-9.
Journal code: DEA. ISSN: 0090-1229.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 199805
EW 19980501

L7 ANSWER 2 OF 24 MEDLINE
AN 1998045878 MEDLINE
DN 98045878
TI A novel strategy for myocardial protection using in vivo transfection of cis element '**decoy**' against NFkappaB binding site: evidence for a role of NFkappaB in ischemia-reperfusion injury.
AU Sawa Y; Morishita R; Suzuki K; Kagisaki K; Kaneda Y; Maeda K; Kadoba K; Matsuda H
CS First Department of Surgery, Institute for Cellular and Molecular Biology, and Fujisawa Pharmaceutical Co, Ltd, Osaka University Medical School, Suita, Japan.
SO CIRCULATION, (1997 Nov 4) 96 (9 Suppl) II-280-4; discussion II-285.
Journal code: DAW. ISSN: 0009-7322.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 199802
EW 19980204

L7 ANSWER 3 OF 24 MEDLINE
AN 1998044827 MEDLINE
DN 98044827
TI Controversies in the cellular pharmacology of oligodeoxynucleotides.
AU Stein C A
CS Department of Medicine, Columbia University, College of Physicians and Surgeons, New York 10032, USA.
NC 60639
SO CIBA FOUNDATION SYMPOSIUM, (1997) 209 79-89; discussion 89-93. Ref: 34
Journal code: D7X. ISSN: 0300-5208.
CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA English
FS Priority Journals
EM 199803
EW 19980304

L7 ANSWER 4 OF 24 MEDLINE
AN 97398383 MEDLINE
DN 97398383
TI In vivo transfection of cis element "**decoy**" against nuclear factor-kappaB binding site prevents myocardial infarction [see comments].
CM Comment in: Nat Med 1997 Aug;3(8):834-5
AU Morishita R; Sugimoto T; Aoki M; Kida I; Tomita N; Moriguchi A; Maeda K; Sawa Y; Kaneda Y; Higaki J; Ogihara T
CS Department of Geriatric Medicine, Osaka University Medical School, Suita, Japan.
SO NATURE MEDICINE, (1997 Aug) 3 (8) 894-9.
Journal code: CG5. ISSN: 1078-8956.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199711
EW 19971101

L7 ANSWER 8 OF 24 MEDLINE
AN 96275790 MEDLINE
DN 96275790
TI The NF-kappaB transcription factor in oncogenesis.
AU Sharma H W; Narayanan R
CS Division of Oncology, Roche Research Center, Hoffmann-La Roche, Inc., Nutley, NJ 07110, USA.
SO ANTICANCER RESEARCH, (1996 Mar-Apr) 16 (2) 589-96. Ref: 74
Journal code: 59L. ISSN: 0250-7005.
CY Greece
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA English
FS Priority Journals; Cancer Journals
EM 199610

L7 ANSWER 9 OF 24 MEDLINE
AN 96200687 MEDLINE
DN 96200687
TI Transcription factor **decoy** approach to decipher the role of **NF-kappa B** in oncogenesis.
AU Sharma H W; Perez J R; Higgins-Sochaski K; Hsiao R; Narayanan R
CS Division of Oncology, Roche Research Center, Hoffman-La Roche Inc., Nutley, NJ 07110, USA.
SO ANTICANCER RESEARCH, (1996 Jan-Feb) 16 (1) 61-9.
Journal code: 59L. ISSN: 0250-7005.
CY Greece
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 199608

L7 ANSWER 12 OF 24 MEDLINE
AN 94019327 MEDLINE
DN 94019327
TI Inhibition of phorbol ester-induced cellular adhesion by competitive binding of **NF-kappa B** in vivo.
AU Eck S L; Perkins N D; Carr D P; Nabel G J
CS Department of Internal Medicine, University of Michigan Medical Center, Ann Arbor 48109-0650.
SO MOLECULAR AND CELLULAR BIOLOGY, (1993 Oct) 13 (10) 6530-6.
Journal code: NGY. ISSN: 0270-7306.
CY United States

DT Journal; Article (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199401

L7 ANSWER 16 OF 24 BIOSIS COPYRIGHT 1998 BIOSIS
AN 98:24283 BIOSIS
DN 01024283
TI Feasibility of **decoy** strategy by targeting the transcription factor **NF-kappa-B** in anti-GBM nephritis.
AU Tomita N; Morishita R; Hashizume M; Yasuba M; Notake M; Fujitani B; Yamamoto K; Lan H; Kaneda Y; Higaki J; Ogihara T
CS Osaka Univ., Suita, Japan
SO 30th Annual Meeting of the American Society of Nephrology, San Antonio, Texas, USA, November 2-5, 1997. Journal of the American Society of Nephrology 9 (PROGRAM AND ABSTR. ISSUE). 1997. 467A. ISSN: 1046-6673
DT Conference
LA English

L7 ANSWER 18 OF 24 BIOSIS COPYRIGHT 1998 BIOSIS
AN 96:6401 BIOSIS
DN 98578536
TI Inhibition of human mesangial cell proliferation by **decoy oligonucleotide** targeting the transcription factor, **NF-kappa-B**.
AU Kashihara N; Maeshima Y; Sekikawa T; Okamoto K; Kanao K; Sugiyama H; Makino H; Ota Z; Yasuda T
CS Okayama Univ. Med. Sch., Okayama, Japan
SO Annual Meeting of the American Society of Nephrology, San Diego, California, USA, November 5-8, 1995. Journal of the American Society of Nephrology 6 (3). 1995. 834. ISSN: 1046-6673
DT Conference
LA English

L7 ANSWER 20 OF 24 CAPLUS COPYRIGHT 1998 ACS
AN 1996:618372 CAPLUS
DN 125:316051
TI Gene therapy of renal diseases
AU Imai, Enyu; Isaka, Yoshitaka; Akagi, Yoshitaka; Ando, Yutaka; Kaneda, Yasufumi
CS Med. Sch., Osaka Univ., Suita, 565, Japan
SO Sogo Rinsho (1996), 45(10), 2299-2305
CODEN: SORIAX; ISSN: 0371-1900
DT Journal; General Review
LA Japanese

L7 ANSWER 21 OF 24 CAPLUS COPYRIGHT 1998 ACS
AN 1995:753502 CAPLUS
DN 123:208768
TI In vivo therapeutic use of **oligonucleotide** cis-element decoys for transcription factor binding
IN Dzau, Victor J.; Gibbons, Gary H.; Morishita, Ryuichi
PA USA
SO PCT Int. Appl., 26 pp.
CODEN: PIXXD2
PI WO 9511687 A1 950504
DS W: CA, JP
RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE
AI WO 94-US12339 941028
PRAI US 93-144717 931029
DT Patent
LA English

=> d 1 2 3 4 8 9 12 [redacted] 18 20 21 ab

L7 ANSWER 1 OF 24 MEDLINE
AB NF-kappaB is a potential target for immunosuppressive therapy. Two methods were evaluated to inhibit NF-kappaB: the antisense (AS) approach in which single-stranded oligodeoxynucleotides (ODNs) bind the mRNA for the RelA subunit of NF-kappaB and the transcription factor **decoy** (TFD) approach in which double-stranded ODNs bind the NF-kappaB protein. AS and TFD inhibited NF-kappaB binding and decreased total IgG and anti-dsDNA antibody production in splenocytes from the BXSB/Yaa autoimmune mouse strain. TNF-alpha expression was reduced by AS and TFD, as were the levels of IL-2. But AS effects did not last beyond 24 h, whereas TFD inhibited cytokine production after 72 h. AS had no effect upon IL-6, while the TFD reduced the secretion of IL-6. Therefore, the suppression of immune response mediators by AS or TFD, through inhibition of NF-kappaB, is substantial. These inhibitors can serve as novel choices for therapy in the treatment of autoimmune disorders. Copyright 1998 Academic Press.

L7 ANSWER 2 OF 24 MEDLINE
AB BACKGROUND: NFkappaB, an important transcriptional factor, has been reported to play a significant role in the coordinated transcription of cytokine and adhesion molecule genes. Therefore, blocking the NFkappaB may attenuate ischemia reperfusion injury in the myocardium. For blocking transcriptional factors, gene therapy, such as cis element "**decoy**," appears to be an innovative and useful therapy. This study aimed to prove the efficacy of cis element **decoy** against NFkappaB binding site for myocardial protection. METHODS AND RESULTS: Rat hearts were transfected with fluorescence isothiocyanate-labeled cis element **decoy** against NFkappaB (NF)-binding site (NF group, n=6) and scrambled **decoy** (SD) group (n=6) by coronary infusion of hemagglutinating virus of Japan (HVJ)-liposome during cardioplegic arrest. Both the NF and SD groups showed marked FITC-staining in the nuclei of myocytes, demonstrating the efficacy of gene transfer into the nuclei of cardiac myocytes as compared with the control group transfected with empty liposomes. After 3 days of transfection, the NF group showed significantly higher percentages of recovery of left ventricular developed pressure (NF versus SD, 87+/-11 versus 54+/-12%) and coronary flow (97+/-16 versus 61+/-15%) than did the control hearts when exposed to ischemia (30 minutes, 37 degrees C) and reperfusion (30 minutes, 37 degrees C). The NF group showed a significantly lower percentage of neutrophil adherence to endothelial cells (38+/-6 versus 81+/-3%) and a lower tissue level of interleukin-8 (109+/-48 versus 210+/-55 ng/mg) than did the SD group. CONCLUSION: The hearts transfected with cis element **decoy** against NFkappaB binding site showed significant improvement in tolerance against ischemia-reperfusion injury in association with the inhibition of neutrophil adherence and tissue IL-8 production. This suggests that NFkappaB plays a significant role in ischemia-reperfusion injury. This method, using in vivo gene transfection of cis element **decoy** against NFkappaB binding site, appears to be a novel and future strategy for myocardial protection.

L7 ANSWER 3 OF 24 MEDLINE
AB Phosphodiester and phosphorothioate oligodeoxynucleotides are polyanions that cannot passively diffuse across cell membranes. Instead, the processes of adsorbtive endocytosis and pinocytosis probably account for the great majority of oligodeoxynucleotide internalization in most cell types. Oligodeoxynucleotides can adsorb to heparin-binding, cell surface proteins. An example of such a protein is the integrin Mac-1 (alpha M beta 2; CR3; CD11b/CD18), a

receptor for fibrinogen which is found on neutrophils, macrophages and natural killer cells. Up-regulation of neutrophil cell surface Mac-1 expression by interleukin 8, arachidonic acid or tumour necrosis factor alpha leads to increased cell surface oligodeoxynucleotide binding and internalization. Binding and internalization can be blocked by both fibrinogen and by anti-Mac-1 monoclonal antibodies. Subsequent to internalization, oligodeoxynucleotides reside in subcellular vesicular structures, i.e. endosomes and lysosomes. However, in the absence of permeabilizing agents, these compartments may be sites of sequestration and the oligomers may be unavailable for antisense activity. At present, controversy surrounds the use of guanosine-rich phosphorothioate oligodeoxynucleotides as antisense agents. We examined the ability of the 24mer antisense rel A (p65) phosphorothioate oligodeoxynucleotide to inhibit nuclear translocation of **NF kappa B** in K-BALB murine fibroblasts. 7-Deaza-2'-deoxyguanosine substitution in the 5' guanosine quartet region demonstrated that inhibition of nuclear translocation could not be due to a Watson-Crick antisense effect. Rather, we favour the explanation that the parent molecule may be a sequence-specific, apatameric **decoy**.

L7 ANSWER 4 OF 24 MEDLINE

AB The transcriptional factor nuclear factor-kappaB (NFkappaB) plays a pivotal role in the coordinated transactivation of cytokine and adhesion molecule genes that might be involved in myocardial damage after ischemia and reperfusion. Therefore, we hypothesized that synthetic double-stranded DNA with high affinity for NFkappaB could be introduced in vivo as "**decoy**" cis elements to bind the transcriptional factor and to block the activation of genes mediating myocardial infarction, thus providing effective therapy for myocardial infarction. Treatment before and after infarction by transfection of NFkappaB **decoy**, but not scrambled **decoy**, oligodeoxynucleotides before coronary artery occlusion or immediately after reperfusion had a significant inhibitory effect on the area of infarction. Here, we report the first successful in vivo transfer of NFkappaB **decoy** oligodeoxynucleotides to reduce the extent of myocardial infarction following reperfusion, providing a new therapeutic strategy for myocardial infarction.

L7 ANSWER 8 OF 24 MEDLINE

AB The NF-kappaB transcription factor complex is a pleiotropic activator that participates in the induction of a wide variety of cellular and viral genes. The active complex is composed of two subunits designated NFKB1 and RelA (formerly called p50 and p65, respectively). Binding sites for NF-kappaB are present in the promoter region of many cell adhesion molecules, cytokines and growth factors. Antisense inhibition of the individual subunits of NF-kappaB exerted differential effects on cell adhesion. Antisense phosphorothioate oligomers to relA but not NFKB1 caused a rapid inhibition of cell adhesion in diverse cell types. Antisense relA oligomers exerted antigrowth effects on diverse transformed cells in vitro and caused a pronounced inhibition of tumorigenicity in nude mice tumor models. Stable transfectants of a fibrosarcoma cell line expressing dexamethasone-inducible antisense RNA to relA also showed inhibition of in vitro growth and in vivo tumor development. In response to inducible expression of antisense RNA, a pronounced tumor regression was seen in nude mice. Use of a "**decoy**" approach to inhibit RelA function directly also caused inhibition of tumor cell growth in vitro and in vivo. Our results indicate that key regulatory molecules such as transcription factors can be selectively targeted for therapeutic intervention in cancer.

L7 ANSWER 9 OF 24 MEDLINE

AB Antisense inhibition of the RelA subunit of **NF-kappa B** transcription factor (but not the NFKB1 subunit) causes pronounced inhibition of tumor cell growth in vitro and in vivo. Inhibition of either subunit, however, results in inhibition of the heterodimeric **NF-kappa B** complex in antisense-treated cells. Either of the subunits of **NF-kappa B** can form homo- or heterodimers with other members of the Rel oncogene family. In an effort to decipher the role of homo- vs heterodimeric NNF-kappa B in regulating tumor cell growth, we have used a **decoy** approach to trap these complexes in vivo. Using double-stranded phosphorothioates as a direct in vivo competitor for homo- vs heterodimeric **NF-kappa B**, we demonstrate that decoys more specific to RelA inhibit growth tumor cell growth in vitro. We demonstrate that RelA, either as a homodimer or a heterodimer with some other members of the Rel family and not the classical **NF-kappa B** (RelA/NFKB1), is involved in the differential growth control of tumor cells. Our results indicate that such transcription factor decoys can be a non-antisense tool to study the function of DNA-binding transcription factors.

L7 ANSWER 12 OF 24 MEDLINE

AB Adhesive interactions between cells are essential for the organization and function of differentiated tissues and organs and are mediated by inducible cell surface glycoproteins. In normal tissues, cell adhesion molecules contribute to immune regulation, inflammation, and embryogenesis. Additionally, they play an important role in a variety of pathogenic processes. Cell adhesion molecule expression can be induced by stimuli known to activate **NF-kappa B**, a ubiquitous transcription factor found in a variety of cell types. To investigate the role of **NF-kappa B** in cell adhesion molecule expression, we treated HL-60 cells with a double-stranded **oligonucleotide** which specifically inhibits **NF-kappa B**-mediated transcription. This treatment resulted in the inhibition of phorbol 12-myristate 13-acetate (PMA)-induced cellular adhesion, morphological changes, and the expression of leukocyte integrin CD11b. In a similar fashion, expression of intercellular adhesion molecule 1 on human endothelial cells induced by PMA was specifically inhibited by the **NF-kappa B antagonist**. We suggest that **NF-kappa B** activation is a necessary event for the PMA-induced differentiation of HL-60 cells and the expression of certain activation is a necessary event for the PMA-induced differentiation of HL-60 cells and the expression of certain adhesion molecules. Furthermore, the inhibition of transcription factor functions by this generally applicable mechanism can be used to define their role in cellular differentiation and function.

L7 ANSWER 16 OF 24 BIOSIS COPYRIGHT 1998 BIOSIS

L7 ANSWER 18 OF 24 BIOSIS COPYRIGHT 1998 BIOSIS

L7 ANSWER 20 OF 24 CAPLUS COPYRIGHT 1998 ACS

AB A review with 16 refs., on the methods for gene transfer to target cells in vivo or ex vivo, merits and demerits of HVJ-liposome method, glomerulosclerosis induced by transfection of TGF-.beta. or PDGF-.beta. gene into rat kidney, and gene therapy for glomerulonephritis by HVJ-liposome method. Antisense **oligonucleotide** of TGF-.beta., **decoy oligonucleotide** for **NF-kB**, gene for decorin (natural inhibitor of TGF-.beta.), gene for TGF-.beta. receptor-IgG Fc chimeric protein, and 15-lipoxygenase gene have

therapeutic pot[redacted]ial for gene therapy of glomerulonephritis. A new gene transfer m[redacted]od using mesangial cell vector[redacted] also introduced.

L7 ANSWER 21 OF 24 CAPLUS COPYRIGHT 1998 ACS

AB Oligodeoxynucleotide decoys are provided for prophylactic or therapeutic treatment of diseases assocd. with the binding of endogenous transcription factors to genes involved in cell growth, differentiation, and signaling, or to viral genes. By inhibiting endogenous trans-activating factors from binding transcription regulatory regions, the decoys modulate gene expression and thereby regulate pathol. processes including inflammation, intimal hyperplasia, angiogenesis, neoplasia, immune responses, and viral infection. The decoys are administered in amts. and under conditions whereby binding of the endogenous transcription factor to the endogenous gene is effectively competitively inhibited without significant host toxicity. The subject compns. comprise the **decoy** mols. in a context which provides for pharmacokinetics sufficient for effective therapeutic use. Thus, a 14-bp double-stranded DNA **oligonucleotide** (5'-CTAGATTTCCCGCG-3'/3'-TAAAGGGCGCCTAG-5') was transfected into vascular smooth muscle cells and shown to effectively abolish the binding of the E2F transcription factor to a specific binding site in serum-stimulated cells. Induction of c-myc, cdc2, and PCNA mRNA expression in response to serum stimulation was markedly inhibited by transfection of the E2F **decoy**, whereas there was no effect on .beta.-actin mRNA expression. Phosphatidylserine/phosphatidylcholine/cholesterol liposomes contg. inactivated hemagglutinating virus of Japan (Z strain) were used to encapsulate the dsDNA decoys, resulting in a more rapid cellular uptake and nuclear concn., and a 100-fold higher transfection efficiency than lipofection or passive uptake methods. Neg. response element (NRE):NRE-binding protein interaction responsible for silencing of renin Ren1 gene expression was also affected by dsDNA decoys in cell line (SCA-9) derived from a submandibular gland tumor.

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=> s nf kappa beta

          9796 NF
          4128 KAPPA
        167222 BETA
L1           1 NF KAPPA BETA
                 (NF(W)KAPPA(W)BETA)

=> s nf kappa b

          9796 NF
          4128 KAPPA
       1134873 B
L2           96 NF KAPPA B
                 (NF(W)KAPPA(W)B)

=> s nf kb

          9796 NF
         12673 KB
L3           62 NF KB
                 (NF(W)KB)

=> s l1 and l2 and l3

L4            0 L1 AND L2 AND L3

=> s l1 or l2 or l3

L5          142 L1 OR L2 OR L3

=> s l5 and (decoy or antagonist)

           783 DECOY
          8579 ANTAGONIST
L6           27 L5 AND (DECOY OR ANTAGONIST)

=> d 1-27
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[IMAGE AVAILABLE]

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